An investigation of the performance of a new Mechanical thrombectomy device using Bond Graph modelling: application to the extraction of blood clots in the middle cerebral artery.

Gregorio Romero*, M Luisa Martinez, Jesus Felez, Gillian Pearce and Neil D Perkinson

Abstract
A number of Thrombectomy devices using a variety of methods have now been developed to facilitate clot removal. We present research involving one such experimental device recently developed in the UK, called a 'GP' Thrombus-Aspiration-Device (GPTAD). This device has the potential to bring about the extraction of a thrombus. Although the device is at a relatively early stage of development, the results look encouraging. In this work, we present an analysis and modelling of the GPTAD by means of the Bond Graph technique; it seems to be a highly effective method of simulating the device under a variety of conditions. Such modelling is useful in optimising the GPTAD and predicting the result of clot extraction. The aim of this simulation model is to obtain the minimum pressure necessary to extract the clot and to verify that, both the pressure and the time required to complete the clot extraction are realistic for use in clinical situations, and are consistent with any experimentally obtained data. It is therefore necessary to consider aspects from rheology and mechanics.

Keywords
Biomedical engineering, Bond-Graph methodology, thrombectomy device.

1. INTRODUCTION

Stroke is a major cause of morbidity and mortality globally. In the UK alone there are 130.000 strokes each year [1]. Even those who survive the initial insult of stroke are often left with residual disability resulting in profound impact on their life style and life expectancy.

Approximately 85% of strokes are caused by a blood clot. Intravenous thrombolysis using alteplase is an effective treatment for acute stroke [2]. However, there are limitations on its use. Due to the risk of haemorrhage it is contraindicated in patients who have undergone recent surgery. It has to be given relatively early after the stroke and it is only licensed for use up to 3 hours post stroke [3], although a more recent study has indicated that it could possibly be used up to 4 to 5 hours. It can therefore only be used in certain patients who have suffered a stroke.

The need to study new medical devices like the one described here means that a computer pre-modelling is required in order to proceed to a better optimization and fine-tuning of the device. The aim of the simulation model that is presented is to obtain the minimum pressure necessary to extract the clot and to check that, both this pressure and the time required to complete the operation are reasonable for potential use in clinical situations, and are in line with experimentally obtained data.

2. THROMBOEMBOLIC STROKES

Stroke occurs as a result of ischemic or hemorrhagic vascular disease. Ischemic stroke has the potential for damage in the penumbral area and in the core, and treatment aims to remove the clot. Methods have included surgery, clot-dissolving drugs, and insertion of thrombus removal devices [4]. During the last decade MTDs have become more widely used.

Major contributing risk factors for stroke include dysrthymias e.g. atrial fibrillation; hypertension, cigarette smoking and transient ischaemic attacks. A hypertensive individual has a four to six fold increased risk of having a stroke than a non hypertensive person, and studies have shown that antihypertensives can decrease the risk of the occurrence of stroke [5].

Atherosclerosis is usually caused by excess levels of LDL circulating in the body, leading to the formation of an atheromatous plaque, which may block the blood vessels and may contribute to the formation of blood clots[5].

Smoking increases the risk of atherosclerosis and may increase the damage to the endothelial wall of blood vessels thus increasing the risk of formation of bloods clots leading to an increased risk of stroke.

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Smokers have an overall relative risk of 1,5 times that of non-smokers [5]; moreover there is a 2,4 greater risk of strokes in heavy smokers (20 or more cigarettes a day). Excess alcohol consumption may affect blood clotting and blood viscosity, in addition to increasing blood pressure. This may in turn give rise to an ischaemic stroke. Other causes of stroke include genetic factors e.g. Fabry's disease and CADASIL (Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy) [6]. Treatments for thromboembolic strokes can be divided into three categories:

- Thrombolytic agents;
- Neuroprotective agents; and
- Endovascular Thrombectomy (Mechanical devices)

Thrombectomy devices have been developed as an alternative means for clot removal. A number of devices using a variety of methods to remove the clot are now available. These include the MERCI clot retriever [7], and, more recently, the penumbral device [8]. Other types of devices include angiography catheters [9] rheolytic catheters (Angiojet) [10], Basket style devices [11] and microsnaring devices [12]. Thrombectomy has associated risks, such as breakage of moving parts, penetration of the arterial wall, and downstream embolisation caused by clot dislodgment [13, 14]. Studies suggest that mechanical embolectomy is most effective in large volume proximal occlusions [15].

Other interventional surgical treatments include endarterectomy which involves surgically removing the plaque in the carotid arteries. This treatment has proved successful [16] but carries a risk of the plaque becoming dislodged during the procedure.

3. 'GP' TAD DEVICE

The 'GP' device (figure 1) [17] is attached to a pump that provides the necessary suction pressure for the clot removal, joined to a very long catheter; the 'GP' TAD is located at the end of this catheter. This device is introduced into an artery in close proximity to the occluding blood clot , and is positioned at a distance of approximately 3 mm from it. Then the suction increased until the clot is extracted. The clot crosses the 3 mm that separates it from the 'GP' TAD and clot capture occurs the device is then removed from the body.

It is currently being developed as a potential thrombus aspiration device through a series of in vitro studies. This device has the potential to be used in relatively small arteries. It has no moving parts and therefore should reduce the risk of breakage in a vessel. Since it does not touch the clot itself and hence it should also potentially reduce the risk of downstream embolisation. Thrombectomy is achieved by aspiration through a catheter in which the 'GP' device is embedded. The internal surface has been mathematically optimized . It is also associated with low forces at the periphery of the device which may therefore reduce the risk of arterial collapse during aspiration of the clot [18, 19, 20]. The 'GP' Aspiration Thrombus Device (TAD) has been modelled using a 3-D FLUENT © simulation software package [21]. In addition, different existing techniques have been considered for the modelling and simulation [22]; some methods which have been considered for their applicability are the Boltzmann flow simulation technique, and finite elements modelling and its implementation in Matlab ©, with 2D or 3D models; or by means of Laplace transformations using Dynamic Motion Solver © software.

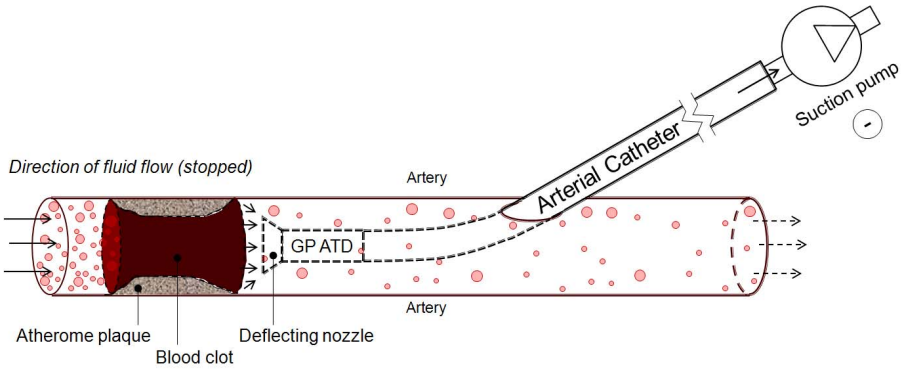

Figure 1. Schematic view of the 'GP' Aspiration Thrombus Device

The objective of this study is to introduce a different method to reproduce the model and it can be used in showing the potential performance of the 'GP' TAD device under different conditions of blood flow, size of blood clot, in a given vessel. The method chosen for the representation and simulation of this model is the Bond Graph technique [23]. Some medical models have been modeled by using this technique [24, 25] and its choice is based on the fact that this technique allows assimilation of the model to an electric circuit made up of resistances, capacitances and inductances.

Therefore, it is possible to obtain the results in a simple way by evaluating flows and efforts that join and connect the components of the model.

4. MODELLING THE 'GP' ATD DEVICE

In this section, we will describe the model used for the simulation considering the different phenomena to define the device; in addition, the values of the parameters used are defined.

4.1. Pump

The pump is the component that creates the necessary pressure to carry out the extraction. It is represented by a variable pressure source (*Se=P*) whose value will increase from zero to 100 kPa, a figure which experience shows to be suitable for carrying out this operation. The time taken to reach 100 kPa is 1 sec., after which time the pressure provided by the pump remains constant.

4.2. Catheter

The catheter is about 1 meter long 1 mm diameter hollow cylindrical plastic tube. It is joined to the 'GP' cylinder of the same diameter and a length of 20 mm. In order to represent both elements, they are considered as several pipe sections bearing in mind the different phenomena that take place in their interior: load (R), the inertia loss (I) and the compressibility of the blood (C) [26].

Linear load loss is due to the friction between the liquid particles and the pipe walls. Due to their being straight pipes, only linear load losses are taken into account. As this pipe is horizontal and of constant cross section in each section, the load loss is reduced to a pressure loss as the fluid advances along the pipe, the loss being progressive and proportional to the length of the pipe. It is represented by a resistance R, and a type 1 junction.

To determine the equation that governs its behaviour, it is necessary to know if the behaviour of the blood flow is laminar or turbulent. This is evaluated by the Reynolds number, giving the following value:

$$\mathrm{Re} = \frac{V \cdot D}{\nu} \approx 1000 < 2200 \qquad (1)$$

Considering a laminar behaviour, the losses can be determined by the following expression:.

$$R_{catheter} = \frac{128 \cdot \eta \cdot L}{\pi \cdot D^4} \qquad (2)$$

where η is the dynamic viscosity of the blood flow, L the length of the pipe section and D its diameter. In each step of the simulation the Reynolds number is checked to be according with this criterion.

Secondly, the flow inertia to be overcome in its movement is taken into account and considering a section with circular geometry it can be modelled with this expression:

$$I_{catheter} = \frac{\rho \cdot L}{\pi \cdot \left(D/2\right)^2} \qquad (3)$$

where ρ is the blood density, L the length of the pipe section and D its diameter.

Lastly, the blood compressibility is included. It acts as a spring producing a decrease in volume when the pressure required for compression is increased. This behaviour is dependent on Bulk's blood coefficient (B) and it is defined by a capacitance C with a type 0 junction, by the following expression:

$$K_{catheter} = 4 \cdot B / \pi \cdot D^2 \cdot L \qquad (4)$$

In case of use a catheter elastic in the longitudinal direction, would be necessary to include the elasticity of the material to take into account the length variation; in this case we are considering a very rigid catheter in the longitudinal direction and it is the reason why we are not considering it.

In the model to optimize, first the pump is positioned then the catheter. Due to its great length it could be partitioned in identical sections including the three previously described phenomena to study the evolution of the pressure loss along the catheter, but due to the linear load loss it can be represented only one time.

4.3. GP

After the catheter, the 'GP' device must be positioned and it can be represented by the same three previous phenomena and with the corresponding values. In addition, due to the artery being located at the end of the 'GP' device, it is necessary to consider the transition between both elements as a pressure loss caused by the difference in diameter of the 'GP' device and the artery respectively and the subsequent variations in flow.

4.4. Deflecting nozzle

The load losses commented below can be represented as a secondary load loss and it can be represented by a resistance considering the difference of both diameters – 'GP' device and artery - with the following expression.

$$R_{nozzle} = 8 \cdot \rho \cdot \xi \cdot \frac{Q}{\pi^2 \cdot D^4} \qquad (5)$$

where ρ is the blood density, Q is the flow which circulates in the section between the end of the 'GP' and the artery, D is the mean diameter between the 'GP' and the artery, and ξ the load loss coefficient.

$$D = \frac{D_{GP} + D_{artery}}{2} \qquad (6)$$

The load loss coefficient ξ is a dimensionless parameter that quantifies the loss produced and depends on the geometry of the conical aperture. Considering the results obtained in figure 2, the ideal value for the design of the 'GP' device diameter is that which corresponds with the same diameter that the artery, in which case it would create zero load loss. In patients it is not possible to use the exact value and considering it to be a narrowing with $D_{GP} = 1{,}5 \cdot D_{artery}$, this value can be considered as being 0,3 in the best case.

In eq. (5) the flow which circulates in the section between the end of the'GP' device and the artery is not constant during the extraction, so to calculate this expression, its value at each instant is considered like the flow of the inertance that represent the 'GP' device (*flow(Igp)*).

4.5. Artery

Such as we commented in section 2, the 'GP' device is positioned at a distance of approximately 3 mm from the occluding blood clot, beginning the suction until the clot crosses the distance that separates both and clot capture occurs. This space is corresponding with the artery located between the end of the 'GP' device and the clot and it can be deformed as following figure shows due to his elasticity. Comparing with the rigid catheter material, in this case we need to include this deformation by adding a compliance to represent it.

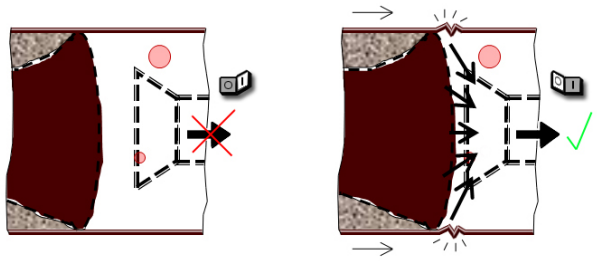

Figure 2. Artery deformation when suction

This piece of artery can be included in the model as another section similar to the catheter or the 'GP' device and it must be defined by the loss of linear load (R), the inertia (I) and the compressibility of the blood (C) in the same way the previous phenomena. In addition and according with the previous comment, it is necessary to insert a parameter that represents the compressibility of the artery, in line with its Young's modulus:

$$K_{artery\_wall} = \frac{E \cdot h}{V_0 \cdot 2 \cdot r_0} \qquad (7)$$

where E is its Young's modulus, h is the thickness of the artery, V_0 is the artery initial volume and r_0 is the artery initial radius.

4.6. Blood clot

Before beginning with the blood clot modeling, it is necessary to think that previous elements has been defined by using a fluid domain; the blood clot element is corresponding with a mass, i.e. a mechanical domain, and it is necessary to change from one to the other domain.

Then, to be able to evaluate the movements and efforts in the clot, as well as to define the physical friction between the clot and the artery adding a Transformer (TF) element. To calculate the value of the coefficient defining this element, the change in the definition of the flow before and after this element is evaluated. Before the TF element, the flow is in the hydraulics domain, while after, it is in the mechanics domain. The coefficient must be determined by evaluating the required change between both domains. Since the equation that relates both flows is $f2=f1 \cdot r$, where $f1=Q1=v1 \cdot A1$ and $f1=v2$, then:

$$r_{TF} = 1/A_1 = 1/\pi \cdot R^2 \qquad (8)$$

where R is the artery radius.

Accurately defining the clot model in order to model it is the most complex part of the modelling. In previous models [27, 28] the clot has been approximated by the authors to a cylindrically-shaped element of [0,5 - 5] cm long, and of a mass that falls between [0,5 - 0,1] gram, connected to the artery wall by using an equivalent spring-damper (figs. 3 and 4), with the same adhesion force every cases. The model has a spring-damper system in parallel to simulate the elastoplastic behaviour of the clot to withstand traction. This representation of the internal conditions of the clot will be maintained in all subsequent models, although they change their name.

Additionally, the clot inertia also supports the bond strength to the artery. To know when the force will be reached in the equivalent spring and, therefore, when the clot movement will begin, we calculated the displacement of the spring when it is subjected to 0,01 N (and other values depending of various parameters) via a typical spring equation. Therefore, only when the spring underwent this displacement did clot movement begin. No clot movement occurred before this value was achieved. This previous model has some difficulties that we should redefine. It focused on the way we simulated the adhesion of the clot to the wall. We think that the simulation of the complete catheter was quite realistic, so we kept it, focusing only on a new model for studying the clot and its adherence to the arterial wall.

Consideration of the adhesion force is relatively complex, and it is challenging to find the spring-damper rate that represents the junction with the wall and we only considered the in-vitro maximum adhesion force. The value of the constants in both the spring and the damper must be

extremely high to simulate a firm anchor to the point of release. Simulating the moment when the clot breaks loose from the wall in the previous model method was very difficult, because the junction to the wall had to allow, clot movement, according to pressure, even if such pressure was not enough to move it in totality.

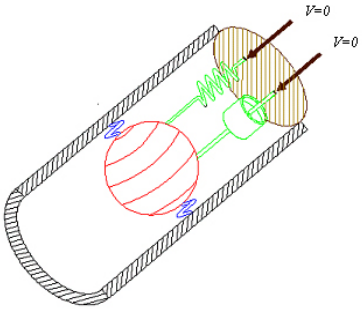

Figure 3. Spring-damper system configuration (F < 0,01 N).

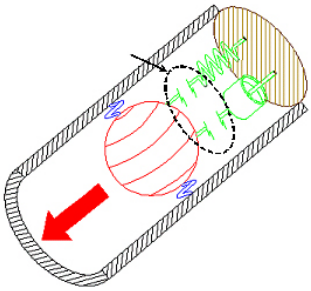

Figure 4. Spring-damper system elimination (F ≥ 0,01 N).

Due to these problems we look for another solution to simulate the clot and its behaviour under pressure. As we have seen before, we kept the partitions represented by inertia and joined by spring-damper system (R_{union}, K_{union}), to simulate the elastic and plastic behaviour of the clot.

To model the junction with the artery, the point of release and the static and dynamic friction, we decided to add to each inertia an effort source that varies depending on the moment of the simulation (Effort source F1 to F5 in fig. 6). Each inertia will suffer a force due to suction, which should be compensated in the model with a force of friction to annul it, while the clot is in the position of static friction. Once we have calculated the flow-effort table of the system, we apply the condition that the stress on the inertia must be zero. The effort source, as we have said before, varies; i.e. when the clot begins its movement, the static friction disappears and the dynamic friction acts in the system. It is much lower that the static friction. We have calculated it by means of the Stokes law for a cylindrical solid:

$$F_{dynamic\_friction} = \frac{C}{8} \cdot \rho \cdot \pi \cdot D^2 \cdot V^2 \qquad (9)$$

where C is the form coefficient for a cylinder, ρ is the blood´s density, D the clot´s diameter and V the velocity of the first partition (*flow(Mclot1)*).

The condition to determine if the clot is attached to the surface is based on the force that the spring suffers between partitions (K_{union}). Hence when $K_{union} \cdot X_{Kunion}$ is higher than the adherence force, the clot releases from the surface. The value of the adherence force was analyzed by C. J. Flannery [29] and we made use of all the necessary data from that. To calculate the adhesion strength he consider a not cylindrical clot that narrows down the middle due to stenosis or atherosclerotic plaque. But this geometry is only valid for the condition of static friction, because, once the clot releases, it recovers a part of its form and again becomes like a cylinder. So, we assume this model and we divided this clot into 5 parts:

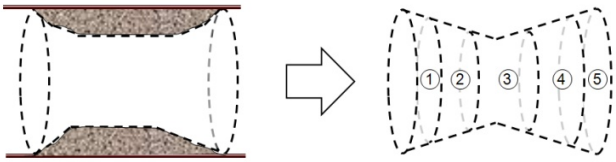

Figure 5. Blood clot 5 parts assumption.

We decided to make these partitions because each one has the same contact area with the artery and the same volume, in order to simplify the calculation of the adhesion force and the mass of each partition. The best option would be to recalculate the contact area and volume of each partition at every moment when it is moving.

As we have defined our clot, we then calculated the adhesion force by means of the platelet adhesion force. And we have obtained from C.J. Flannery the next equation:

$$N^o\ platelets\ per\ area = fp \cdot \frac{SA}{MPA} \qquad (10)$$

where *fp* is the % of platelets in the clot, *SA* is the surface in contact with the artery and *MPA* the Mean Platelet Area.

Once we have the number of platelets in contact with the artery and the force/platelet, we can calculate the adhesion force of each partition and obviously of the entire clot. In this way we would have different adhesion forces depending on the form and size of the clot, and we can reference those to the artery diameter and the percentage of occlusion.

$$F_{adherence} = N^o\ platelets \cdot F_{adhesion\_platelets} \qquad (11)$$

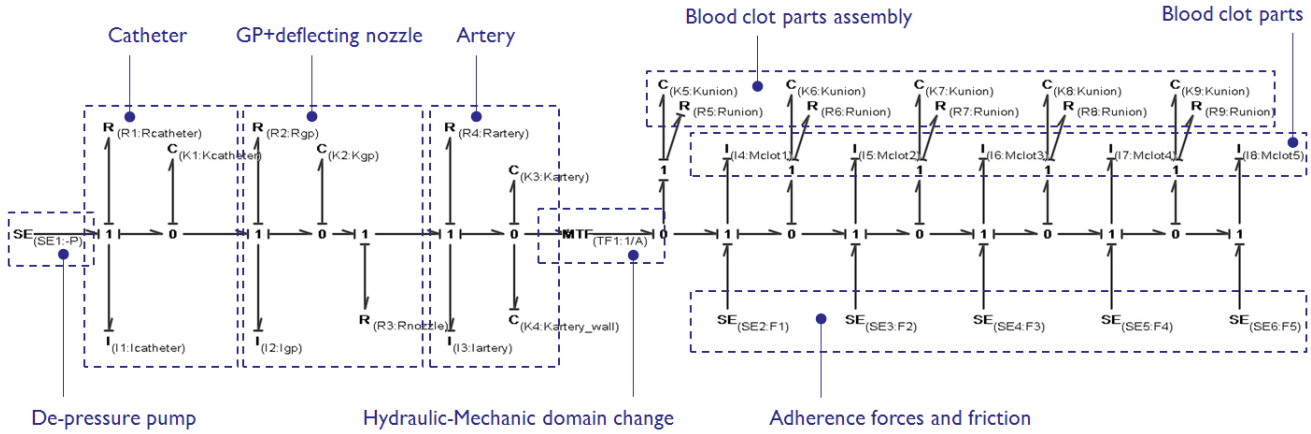

Figure 6. Modelling the full device components and blood clot by Bond Graph technique.

5. RESULTS

The fundamental object of this study consists in determining and optimizing the minimum pressure required for the extraction of a blood clot depending on the clot size. To do this, by varying the values of the pressure source and clot length, the movement of the clot and the time required for its extraction are measured, thereby obtaining the optimum minimum pressure.

The parameters that define almost completely elastic-plastic behaviour of the clot and its resistance to breakage are the constants K_{union} and R_{union} of the spring-damper systems in parallel that are among the partitions of the clot and that characterize the clot in the stretch, when it suffers the suction before it is detached from the wall.

To find the value of the K_{union} parameter, Savushkin [30] analyzes the stiffness of the clot and the breaking strength. We considered that the values are valid, since the parameters of the experiments described fall within our range, and therefore we can assume that K_{union} = 3,41 ± 1,5 N/m. We took the minimum value 1,91 N/m to study the system in the worst possible conditions. We have also find in the referred work the rupture force necessary to break the clot, so we included it also in our simulation ($F_{rupture}$ = 209 ± 73 $\cdot 10^{-3}$ N).

Corresponding with the R_{union} value, Pennati et al. [31] described the values for the viscosity of the blood and the clot that they use in their model; due to the viscosity of the clot; we can assume that R_{union} = 0,035 kg/m·s.

The aim of this simulation is to compare extraction of a clot into the Middle Cerebral Artery (2.5 mm) considering different clot lengths (1 cm up to 5 cm each case) and 70, 80, 90 and 100% of occlusion.

The size of the artery determines the size of the 'GP' TAD used to remove the clot. In the study the used size is 1 mm of diameter for the catheter and the 'GP' device.

To carry out the model validation, the values of the parameters used in the simulation are listed in following table.

Pressure	[0, -100] kPa
Blood Density (ρ)	1060 Kg/m^3
'GP' length (L)	0,02 m
Artery Young modulus (E)	2,8·10^9 N/m
Artery thickness (h)	0,1 mm
Artery length (La)	3 mm
Artery diameter	2,5 mm
Blood Viscosity (η)	0,0035 m
Kunion	1,91 N/m
Runion	0,035 N·s/m
Catheter length	1,0 m
Catheter diameter	1,0 mm
Clot length	[1,0 - 5,0] cm
'GP' diameter	1,0 mm
% of platelets in the clot - fp	0,96
Mean Platelet Area - MPA	5,31·10^{-6} mm^2
Fadhesion_platelet	32·10^{-9} N
Occlusion	[70, 100] %

Table I. Model parameter values.

The existence of different clot lengths will affect the mass of the clot being removed (up to 1 gr.) and increase the time taken for clot removal as the value of the clot length increases. It has been found, that the greater the rigidity of the clot, the shorter the extraction time. This factor is also related to the viscosity and composition of the clot that will vary in each case. Therefore, for this study we have determined the critical values possible, making the assumption that there are 96% platelets in the clot, which yields a fairly high bond strength, which gives us an idea of the maximum pressure needed.

Following figure show the movement of each blood clot part taking into account the increasing value of the suction pressure in the first second according with the comment given at section 4.1. Such as it is possible to analyze, one time the force over one partition is greater than the adhesion force in it between blood clot and artery, the adhesion is broken and it begin the movement; additionally, it is possible to look that the velocity when one section is detached is higher due to all the force is applied over lesser partitions.

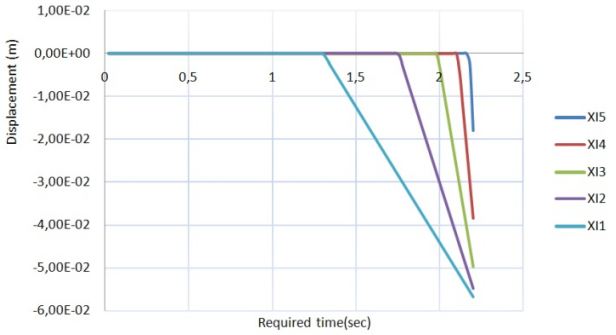

Figure 7. Movement of each blood clot part.

The breaking strength of the clot, is considered, and is taken to an accuracy of an order of magnitude. With this data, it is found that in cases L_{clot} = 5 cm and occlusion percentages 80-100%, there is a danger of rupture prior to complete clot removal, which would mean the failure of the process. Because when the clot breaks up, flow channels appear, and the blood begins to circulate through them. Consequently it is no longer possible to extract the clot. Therefore, it is taken as a benchmark, to call attention to the danger of rupture cases.

Simulating a range of pressures from 0 to -100 kPa, we can observe how it means different times or impossibility to proceed with the extraction of the blood clot due to the adhesion force. The following figures show the time it takes to move the clot 3 mm (first part), which is the distance that is maintained during the extraction to the 'GP' TAD.

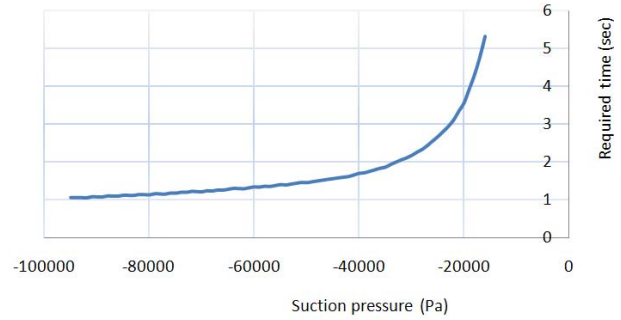

Figure 8. Suction pressure Vs required time (100%, 1cm).

In figure 8 we can observe the different times to extract a blood clot in a 100% occlusion case and 1 cm blood clot. Although our first study was the obtaining of the minimum necessary pressure, we observe clearly the exponential tendency and values below 15 kPa for the suction pump cannot be used every case due to the increasing required time; in this case, the necessary time to detach the first part and be possible the extraction pulling the catheter by hand is about 5 seconds.

Following figures shows the comparison between 70% and 100% of occlusion. It is possible to look how the blood clot length have a very important influence over the required time because it means more inertia due to the mass and more contact area, that is, more adhesion force. For example, in cases of having a 70% occlusion and 4 cm blood clot length we would need 79 secs applying a minimum suction pressure of -35 kPa; however, we would need only 20 secs applying a suction pressure of -39 kPa. In cases where the patient has not time to do it, this pressures and times show a very important difference.

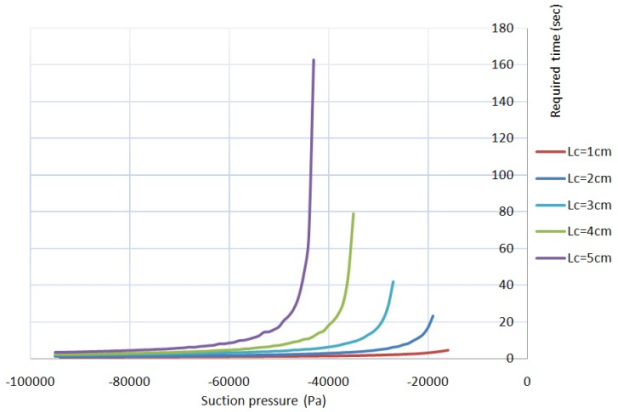

Figure 9. Result for 70% occlusion and different lengths

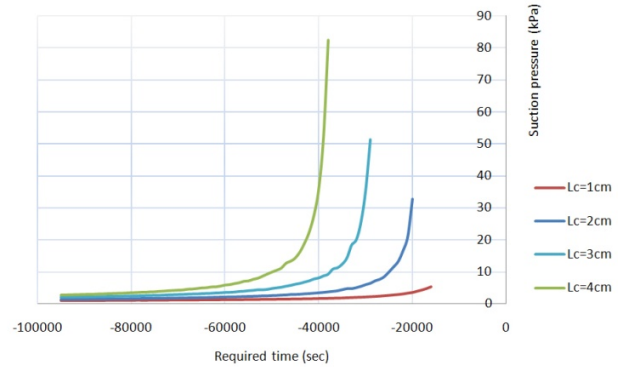

Figure 10. Result for 100% occlusion and different lengths

In the case of having a 100% occlusion and 5 cm length the blood clot would be broken and some extractions would be necessary to do; in the case of 70%, important differences exist in the required time if we use -53±1 kPa.

Comparing the different occlusion percentages, in the figure the lines corresponding with the 70% occlusion are always below the ones for 100% occlusion. That is reasonable because we have more adherence area for a 100% occlusion; hence more adherence force and therefore we need more time to extract the blood clot. However we can observe that for 1 cm length both lines are very similar; that is because for such small clots the difference of adherence force due to the surface is not so significant as in the other cases.

On the other hand, studies demonstrate that the artery has a resistance of 750 mmHg, which is equivalent to 100 kPa. This has been analyzed in the model and the pressure in this area rises to 4-5 kPa in the worst case, there being a wide danger margin for rupture of the artery.

6. CONCLUSIONS

For this model we have focused primarily on the configuration and simulation of the clot, which was still in an early phase of study. We have studied the formation, composition and shape of different blood clots in different cases, trying to find some general parameters that could define their behaviour reliably. In particular we have studied the influence of composition, form and some parameters that directly affect the adhesive force that holds the clot to the arterial wall.

But the most significant inclusion in the model, and therefore the one that provides greater reliability to the model has been the change in the approach regarding the internal structure of the clot and its adherence to the wall. The clot is broken down into smaller partitions that are joined together by a spring-damper system, which represents the elastoplastic behaviour of the clot, and whose parameters are the stiffness and viscosity

To develop the condition of adherence to the wall, detachment and subsequent movement of the clot with viscous friction, it was decided to approach the action-reaction, so that the clot would remain at rest by a frictional force of equal value, but in the opposite direction, to the one associated with each partition until the condition for clot movement is fulfilled. For clot movement to occur, the frictional force changes to the corresponding value for the simulation of dynamic friction. This friction is calculated by Stokes law, and considers the motion of a solid cylinder within a viscous fluid.

With this approach, conditions and parameters, we can obtain a fairly accurate simulation of the behaviour of a blood clot attached to an artery wall. This is evidenced by various simulations and we can demonstrate how each partition is involved in the model.

The influence of the composition, stiffness, viscosity and geometry of the clot on the clot extraction time can be seen. The effect of increasing the extraction time factors such as the percentage of platelets, the viscosity, the length and decreasing of the rigidity of the clot can be modelled. The results obtained from modelling are consistent with those observed in in-vitro measurements.

So we considered that the model is valid and can be used for different cases now and in the future, as more experimental data become available.

It is therefore concluded that the model fits in reality and can be extrapolated to different models and cases, due to the flexibility and approach of the model under various conditions. This model may be used more generally as opposed to the previous models we developed which could only be used in particular conditions.

The main objective of the development of this simulation model was to obtain the range of pressure required to perform the clot extraction and to check that this pressure together with the time required to complete the operation are reasonable and within acceptable clinical boundaries for eventual potential use of the 'GP' TAD in a clinical setting. We also confirm that the results are similar to experimentally obtained data in vitro. These studies allow optimization of the device to assist in possible future use of this device in patients with thrombosis.

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